4:30

NON-SUSTAINED VENTRICULAR TACHYCARDIA ON HOLTER IS NOT OF PROGNOSTIC SIGNIFICANCE IN ASYMPTOMATIC PATIENTS WITH HYPERTROPHIC CARDIOMYOPATHY

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Non-sustained ventricular tachycardia (VT), recorded in >25% of patients with hypertrophic cardiomyopathy (HC) during 48-hour Holter monitoring, has been associated with a 3 to 4 fold increased incidence of sudden death (SD): an 8% annual mortality that is unaffected by conventional antiarrhythmic (AA) therapy but eliminated entirely by amiodarone. These conclusions were however, based on studies that included patients with syncope as well as asymptomatic patients. In contrast, we have reported a high incidence of SD in HC patients with VT who receive amiodarone. To assess the prognostic significance of VT on Holter, we studied the relation of [illegible]cal, hemodynamic, and electrophysiologic (EP) variables to long term outcome in 207 HC patients using multivariate logistic analysis. VT on Holter was present in 105 (51%) patients. Of these, 32 were asymptomatic [17 received no therapy, 9 type 1a AA drugs (5 for atrial arrhythmias), and 6 Ca^{++} or ß blockers for angina].

Fourteen SD occurred during follow up period of 26 ± 18 months (maximum, 7 years). Significant independent predictors of poor outcome were 1) inducibility of sustained VT at EP (ß=3.1, p<0.0001), and 2) a history of cardiac arrest or syncope (ß=2.9, p<0.001). Notably, VT on Holter did not significantly affect outcome and importantly, none of the asymptomatic patients with VT on Holter died suddenly or had syncope.

Conclusion: VT on Holter does not identify HC patients who are at risk of SD, and 2) asymptomatic patients with VT on Holter have an excellent prognosis. Potent AA drugs e.g. amiodarone are unwarranted in this subgroup and may cause malignant arrhythmias to emerge resulting in SD.

5:00

ELEVATED MYOCARDIAL CATECHOLAMINES IN HYPERTROPHIC CARDIOMYOPATHY: AN ARRHYTHMOGENIC SUBSTRATE?

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To elucidate the clinical significance of increased myocardial norepinephrine (MNEC) and epinephrine concentration (MEC) in pts with hypertrophic cardiomyopathy, we investigated their correlation with 48 hour ambulatory ECG arrhythmic parameters. The myocardial catecholamine concentrations in LV endomyocardial biopsies of 34 pts (74% males, aged 32-65 years) were assessed using catechol-O-methyl-transpherase radioenzymatic method.

Using ANOVA statistical analysis, a consistent difference could be shown between MNEC of pts with few (Lown grade [LG] 1,2,3) and with serious ventricular arrhythmias (LG 4,5) (Fd=222.1, p<.0.01). MNEC was significantly higher in pts with LG 5 arrhythmias (927 ± 18 ng x g^{-1} fresh tissue) than in group with LG 1 (653 ± 42 ng x g^{-1} fresh tissue). A similar significant difference was found for MEC (Fd=127.2, p<.0.01), which was demonstrated to be markedly higher in pts with LG 5 (108 ± 5.4 ng x g^{-1} fresh tissue) than in LG 1 (80 ± 5.7 ng x g^{-1} fresh tissue) arrhythmias.

These results suggest that elevated myocardial catecholamines could be an important link in the genesis of serious arrhythmias in hypertrophic cardiomyopathy.

4:45

CIRCADIAN VARIATION IN THE OCCURRENCE OF SUDDEN DEATH IN HYPERTROPHIC CARDIOMYOPATHY

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Several prior investigations have shown a circadian distribution in the occurrence of sudden death (SD) in patients with coronary artery disease. SD is also an important feature of the natural history of hypertrophic cardiomyopathy (HCM). Consequently, this retrospective study was undertaken to determine whether SD in HCM occurred with a particular frequency throughout the day. Precise hour of SD could be ascertained in 84 patients with HCM evaluated by our institution; ages were 8-59 years (mean 29) and 54 (65%) were male. SD did not occur uniformly throughout the day, but rather was distributed in a bimodal pattern (p=0.01; 2-harmonic regression model); a disproportionate number of SD (36 of 84, 43%) occurred in a peak, early in the day, 7 am to 1 pm. A second peak occurrence of SD was evident in the early evening between 7 pm and 10 pm (18 of 84, 22%). In contrast, SD occurred infrequently during sleeping hours of the night and in mid-afternoon. This periodicity in the occurrence of SD was not influenced by cardioactive medications, age, gender, day of week, or month of year. However, patients who died during moderate/severe exertion did so more commonly during those peak morning and afternoon hours for SD. In conclusion, sudden death in HCM demonstrates a bimodal pattern of circadian variability over the 24-hour day, remarkably similar to that described in ischemic heart disease, with two peaks of occurrence, in the morning 7 am - 1 pm and in early evening 7 - 10 pm. These findings suggest that temporally related physiologic changes may play a role in sudden death in patients with HCM.

5:15

LASER MYOPLASTY FOR HYPERTROPHIC CARDIOMYOPATHY: USE OF A THERMOCOUPLE TO CONTINUOUSLY MONITOR LASER-FIBER CONTACT WITH MYOCARDIUM AND FOR DOSIMETRY.

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We investigated the use of a catheter mounted thermocouple(TC) to monitor catheter(C) positioning and dosimetry in the performance of Nd-YAG laser(L) myoplasty. Probe was mounted on a fixation wire .009" in diameter. Probe temperature(T°) is instantaneously transmitted to a monitor which displays an LED readout. In vitro studies demonstrated that T° rose linearly with duration of exposure(EXP) and power when the L fiber(F) was in contact with the myocardium(M) and corresponded to incremental M necrosis. T° in excess of 100°C were associated with extensive charring of M and crater formation at the site of F contact. We then applied these findings in vivo in a canine model. A guiding C was advanced retrograde to the left ventricle and positioned fluoroscopically with the tip perpendicular to the M. The TC-fixation wire was implanted 2-3 mm outside the guiding C, into the M. A 400u F was then advanced and postioned with its tip effacing the tip of the guiding C. L EXP was begun while T° was continuously monitored via the TC. If the T° did not rise immediately upon beginning the EXP, the C was repositioned until an appropriate T° rise occurred. When T° approached 100°C, exposure was interrupted to allow cooling to occur. Using this method, a total of 12 consecutive successful exposures were performed, all resulting in significant necrosis, ranging from 6x7x8mm to 6x25x30mm in size and without charring or crater formation. Conclusions: 1) C mounted TC can be used successfully in vivo to monitor laser interventions and 2) T° changes at the laser-tissue interface can be used to confirm F postitioning and for on-line dosimetry.